Psychosocial factors underlying ethnic disparities in diabetes outcomes

by

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Introduction

"The physician sets about his task with healthy mind and healthy body…[and] the patient knows neither what he is suffering from, nor the cause….[nor] the outcome of his present state…. Surely it is much more likely that the physician gives proper orders, which the patient not unnaturally is unable to follow."

-Hippocrates, "The Art," c. 5th century BCE

Diabetes mellitus (diabetes) is a highly prevalent metabolic disorder that afflicts 9.3% of the US population (29.1 million people)[1], and 382 million people worldwide.[2] In 2012, diabetes was responsible for 1.5 million deaths worldwide, making it the 8th leading cause of death in the world.[3] This figure has increased since 2000, when diabetes was responsible for 1 million deaths.[3] Untreated or inadequately treated diabetes leads to chronic high blood glucose (hyperglycemia), which can cause many adverse effects, including intensified thirst, frequent urination and fatigue. Much more concerning are the potential long term complications such as blindness, peripheral nerve damage, kidney damage, large blood vessel disease, and limb loss. Diabetes accounts for 44% of all new cases of kidney failure and increases the risk of heart disease death and stroke by 2-4 fold in adults.[1] Additionally, 60% of people 20 years and older who need non-traumatic lower-limb amputations also have diabetes.[1] Individuals with Type 2 diabetes have a decreased life expectancy relative to non-diabetic individuals with similar characteristics.[4] In 2012, the ADA estimated that diabetes care cost the U.S. $245 billion, $176 billion of which was from medical expenditures and $69 billion from reduced productivity.[5] The same report stated that on average, individuals with diabetes incur 2.3

times more yearly medical expenses than individuals without diabetes.[5] In light of these facts, it is clear that reducing the morbidity and mortality burden from diabetes is an important public health issue.

Endocrinologists generally recognize three main types of diabetes: Type 1 diabetes, Type 2 diabetes, and gestational diabetes. All three types of diabetes are disorders of metabolism due to disturbances in the function of the hormone insulin. Insulin activates cellular glucose uptake and increased glucose metabolism. It works antagonistically with the hormone glucagon to regulate blood glucose levels. All three types of diabetes are characterized by the inability of cells to take up glucose from the blood, either due to a deficiency of insulin or insensitivity to its effects. Type 1 diabetes is an autoimmune disorder in which the beta cells (insulin-producing cells) of the pancreas are targeted for destruction by the immune system, leading to a systemic insulin deficiency. Type 1 diabetes comprises only 5-10% of all diabetes cases[1], and is thought to result from a combination of environmental and genetic factors. Type 1 diabetes is typically diagnosed in children and young adults. Gestational diabetes is a temporary condition experienced by about 18% of pregnant mothers, usually beginning during the third trimester and resolving after delivery.[6] Although gestational diabetes is temporary, mothers with gestational diabetes have an increased future risk for developing Type 2 diabetes. Type 2 diabetes occurs when cells acquire resistance to insulin, which often leads to a halt in insulin production by the pancreatic beta cells. Type 2 diabetes accounts for 90-95% of diabetes cases[1] and has a genetic- and lifestyle-based etiology. Obesity, poor nutrition, lack of exercise, old age and stress are each associated with the development of Type 2 diabetes. In addition, the onset of Type 2 diabetes occurs much

more gradually than that of Type 1 diabetes, which has an acute onset marked by a characteristically rapid increase in blood glucose.[7] The focus of this thesis will be on Type 2 diabetes.

Diabetes is most frequently diagnosed by testing for elevated blood glucose. There are several different blood glucose tests; fasting plasma glucose (FPG) focuses upon the plasma glucose level after a 12-14 hour fast. A more detailed characterization of blood glucose is achieved via the oral glucose tolerance test (OGTT), in which a standard dose of glucose is administered and blood glucose is measured at baseline and at 30 minute intervals until 2 hours later, which tests the body's ability to clear glucose from the blood. Finally, the percentage of glycosylated hemoglobin (HbA_{1c}) summarizes average blood glucose concentration over a period of 2-3 months. In 2006, the World Health Organization (W.H.O.) defined diabetes as having either FPG $\geq$ 7.0 mmol/l and/or OGTT $\geq$ 11.1 mmol/l with diabetes symptoms.[8] In 2009, diabetes was redefined as having $HbA_{1c} \geq 6.5\%$, which is much more practical in that patients do not have to undergo a fasting or lengthy test. These definitions apply to both Type 1 and Type 2 diabetes.[8]

Treatment of Type 2 diabetes requires individuals to be very involved in their own chronic illness self-management. Generally, initial Type 2 diabetes treatments utilize diet and exercise to lower blood glucose. If executed properly, diet and exercise alone can often return the body to a normoglycemic state and prevent the progression of diabetes. The ADA recommends that individuals with Type 2 diabetes derive most or all of their dietary carbohydrates from whole grains, legumes, fruits and vegetables.[9] The same report also recommends limiting saturated fat intake to less than 7% of total daily

calories, limiting salt intake, and eating fish two times per week. When diet and exercise recommendations alone fail to effectively reduce blood glucose levels, then typically Oral Hypoglycemic Agents (OHAs) are prescribed. OHAs are drugs that lower blood glucose through various mechanisms. For example, biguanides (e.g. metformin), increase the liver's ability to respond to insulin; α-glucosidase inhibitors (e.g. acarbose, miglitol) decreases glucose absorption in the small intestine; sulfonylurea derivatives (e.g. glyburide, glimepride) increase insulin release from the pancreas. If OHAs fail to effectively manage blood glucose, then patients are typically prescribed exogenous insulin. Self-injected or inhaled insulin is always required to treat Type 1 diabetes and is used to treat individuals with poorly controlled Type 2 in combination with OHAs.[10] Individuals using insulin need to frequently self-monitor their blood glucose levels, using a portable personal device known as a glucometer, in order to determine the appropriate insulin dose needed for their current blood glucose and anticipated carbohydrate intake/expenditure. In addition, physicians also usually recommend that people with diabetes regularly monitor themselves for certain symptoms of long-term complications. For example, diabetes can lead to nerve and blood vessel damage in the feet, so it is important that diabetes patients check their feet daily for tissue damage to help prevent the development of non-healing ulcers. Finally, because diabetes can lead to blindness by decreasing blood supply to the retina, the ADA recommends that diabetes patients see an ophthalmologist for annual dilated eye examinations.[11]

While Type 2 diabetes (hereinafter referred to as "diabetes") takes a large toll on the entire population, there are significant racial disparities in diabetes outcomes and diabetes care. In 2011, diabetes was 1.7 times more prevalent among African Americans

than Caucasian Americans.[12] In addition to the disproportionately high prevalence of diabetes among African Americans, African Americans were also 2.2 times as likely to die from diabetes as Caucasian Americans in 2010.[13] African Americans with diabetes also experience an increased rate of diabetic retinopathy[14], are about 4 times more likely to develop kidney disease[15], and have on average 1.7 fewer quality-adjusted life-years (QALYS) than Caucasian Americans with diabetes.[16] It is thought that this disparity stems from a variety of societal, cultural and biological factors.

Several efforts have been made to specifically improve diabetes care for African Americans. In a 2012 review article[17], Betancourt *et al.* described three types of relatively successful recent efforts: 1) community-based efforts, which involve fellow community members, both with and without diabetes, take an active role in helping patients understand diabetes care and set goals for diabetes self-management; 2) distributing health information technology, such as personal electronic devices as a means to communicate with patients about their diabetes care; 3) multifaceted, systemic interventions that make diabetes education more culturally appropriate and/or improve the cultural awareness of physicians treating African American patients. Each of these efforts have been tested in multiple studies and have been shown to be effective in reducing HbA1c of African Americans with poorly controlled Type 2 diabetes.[17]

In spite of these efforts, vast disparities in diabetes treatment outcomes remain. Therefore, a fresh perspective on the causes of these disparities could help policy makers and health care providers develop better interventions or rework existing interventions to help eliminate these disparities. Several hypotheses have been proposed to explain this inequity. One hypothesis is that African Americans have certain genetic factors that lead

to a predisposition to insulin resistance or obesity. Additionally, most peer-reviewed research on the efficacy of different diabetes medications studies a disproportionately high number of Caucasian populations, and therefore may not generalize well to African Americans. Because of this, it is also possible that unidentified, genetically based differences in the way African Americans respond to certain OHAs could be contributing to poor diabetes control among African Americans.[18] Genetic factors have also been theorized to contribute to the increased risk of diabetes-related complications in African Americans.[15]

Differences in obesity, income and education levels could also possibly explain the disparity in diabetes outcomes. Obesity is one of the strongest risk factors for the development of Type 2 diabetes, along with many other conditions. African American cultural dietary practices, particularly in the southern regions of the US and among lower SES individuals, commonly consist of high fat, high carbohydrate meals, which can lead to obesity.[19] In the U.S., the age-adjusted obesity rate among African Americans is 47.8%, compared to 32.6% among non-Hispanic whites.[20] Additionally, in 2012, the U.S. Census reported that the average annual income for African Americans was $33,321, almost half of the average annual income for non-Hispanic whites ($57,009).[21] In 2012, the percentage of individuals without health insurance was 24.9% for annual household incomes of less than $25,000, compared to only 7.9% for annual household incomes over $75,000.[21] Moreover, a lack of financial resources can limit access to healthy food and places to exercise.[22] The ethnic disparities in income and obesity in part explain the disparity in diabetes outcomes and care quality.

However, even after accounting for factors like health care access, socioeconomic status and obesity, African Americans are still found to have poorer diabetes control and to receive poorer quality diabetes care than Caucasian Americans.[23] This could mean that something about the experience of being an African American unrelated to eating habits or health care access is associated with poor diabetes control. One concerning but plausible possibility is that African Americans receive worse quality diabetes care due to discrimination in clinical settings by providers. African Americans with diabetes are less likely to have HbA1c measurements, lipid testing or eye appointments than white patients with the same health care access.[18] Similarly, African Americans are less likely to get flu and pneumonia vaccines from their primary care physicians than white patients.[15] African American patients are also 1.7 times as likely to have ER visits that result in discharge without seeing a physician.[24] Racial biases are manifest in the choices physicians make about their African American patients and the poor communication between physicians and African American patients. This may explain why African Americans have lower adherence to diabetes medications, dietary and exercise recommendations, and other self-management behaviors like self-monitoring of blood glucose, which leads to poorer glycemic control, higher incidence of diabetes, and increased diabetes-related complications. This hypothesis, along with the genetic theories, may explain the additional risk associated with being African American that is unexplained by SES, obesity and health care access.

This thesis will attempt to examine two of the theories described above about ethnic disparities in diabetes outcomes, along with a novel, third theory which will be discussed in detail below. These are that: 1) Poor patient education and poor

communication between health care providers and African American patients leads to decreased adherence to recommended diabetes self-management behaviors, 2) Obesity, physical inactivity, and high fat, high carbohydrate diets are more common among African Americans and are all associated with increased rates of Type 2 diabetes, and 3) Empowerment, or the personal sense of self-efficacy and internal locus of control over one's diabetes, is lower in African American individuals, which could lead to poorer diabetes self-management. The topic of medication adherence and the concept of patient empowerment as they relate to ethnic disparities in diabetes outcomes will be discussed further below.

Adherence

> To repeat the opening sentiment expressed by Hippocrates in "The Art":
> "The physician sets about his task with healthy mind and healthy body…[and] the patient knows neither what he is suffering from, nor the cause….[nor] the outcome of his present state…. Surely it is much more likely that the physician gives proper orders, which the patient not unnaturally is unable to follow."

This quote describes central philosophies about the necessity of medicine and the process of diagnosing and treating patients. Even 2500 years ago, poor patient adherence limited the effectiveness of a physician's recommendations. More surprising than that, however, is the idea that patients and healers hold a joint responsibility to find a treatment plan that is both effective and feasible. It was not until the 1980s that this idea resurfaced again in public health literature.[25,26] Consider how far our knowledge of physiology and pathology

has improved since ancient Greece, where it was common to prescribe the ingestion of heavy metals or poisonous herbs for various diseases; on the other hand, our view of the role of patient adherence in medicine doesn't seem to have progressed much since Hippocrates' time.

Patient adherence was defined by the W.H.O. in 2003 as "the extent to which a person's behavior – taking medication, following a diet, and/or executing lifestyle changes, corresponds with the agreed recommendations from a health care provider"[27]. This definition emphasizes the patient's active role in their self care and takes into account the long-term self care required for chronic conditions like diabetes, congestive heart failure and HIV/AIDS, as opposed to acute conditions. "Adherence" has replaced "compliance" as the preferred terminology because "adherence" emphasizes dialogue between patient and provider, whereas compliance connotes the lack of a choice and/or involvement in following a health care provider's recommendations.

Adherence has been studied as a function of various regimens, interventions, and diseases since the 1950's. It is estimated that overall treatment adherence for chronic conditions averages around 60%.[28] Because it is associated with the development of costly complications and reduced productivity, nonadherence is estimated to cost upwards of $100 billion each year in the U.S.[28] Nonadherence is often viewed in an overly simplistic manner as resulting from a lack in the self-discipline, will power or concern of the patient. However, others suggest that nonadherence is a deliberate, rational choice related to the patient's goals and quality of life, while others point out the possibility of a fairly passive process, such as "forgetting." Nevertheless, even if a patient autonomously decides to reject the recommendation of a health care provider for rational

reasons, preventable costs may still be incurred in the form of intensified medical care and reduced work productivity due to the development of otherwise preventable complications.[29] Poor medication adherence contributes to many complications in patients with chronic heart failure and can lead to a pattern of recurring hospital admissions[28] and ultimately death in some cases.

Adherence to recommended diabetes medication regimens is highly important for maintaining good glycemic control. The first recommended measure to prevent diabetes progression and onset is to develop healthy eating habits and increase physical activity. As described above, OHAs are prescribed when diet and exercise are not sufficient to manage high blood sugar, which can often be a result of poor adherence to provider's recommendations about diet and exercise. OHA nonadherence in Type 2 diabetes leads to poor glycemic control, and if nonadherence is not recognized as the reason, self-injected insulin is often prescribed. Nonadherence to either treatment often leads to hospitalizations and other serious complications caused by hyperglycemia. Thus, poor adherence to diabetes treatment regimens is a large determinant of future treatment outcomes.

The literature remains unclear about whether or not ethnic disparities exist for adherence to diabetes treatment regimens and whether they are significant enough to explain disparities in glycemic control. A study by Adams *et al.* (2008) reported that while African Americans in their population of study had significantly lower OHA adherence than non-Hispanic whites (72% vs. 78%), accounting for this disparity in medication adherence was not sufficient to explain higher HbA1c levels among African Americans.[30] However, in 2004, Pladevall *et al.* found that African American ethnicity

was no longer a significant predictor of HbA1c when metformin adherence was accounted for.[31] Another study by Martin *et al*. (1995) found no significant ethnic differences in adherence to diabetes management behaviors.[32] Egede and Dagogo-Jack also reported that there were no ethnic differences in adherence to diabetes self-management behaviors, with the exception of the practice of monitoring one's own blood glucose, for which African Americans had a 12% lower rate of adherence than Hispanic and white patients.[15] In summary, there have been several conflicting results across studies as to whether a racial gap in diabetes self-management behavior adherence exists and whether it explains the ethnic disparity in glycemic control.

One explanation for poor diabetes medication adherence is poor patient-provider communication.[33] It is important for physicians and other health care providers to assess their patients' comprehension of diabetes care information and treatment regimens[33], and also to ask about medication adherence in a non-judgmental manner.[34] Negatively phrased or leading questions about patients' medication adherence might induce feelings of shame within patients[34], thus making it difficult for providers to detect non-adherence and work to improve it. Racial concordance (whether the racial identity of the patient aligns with that of the provider) is an important factor in patient-provider communication, which can in turn have an influence on medication adherence. A study by Schoenthaler *et al.* (2012) examined the relationships between racial concordance, communication quality and medication adherence with a population of female, African American patients with hypertension.[35] This group found that non-collaborative communication with a Caucasian physician had significant negative impacts on medication adherence, while poor communication with an African American physician had no impact on subsequent

medication adherence.[35] Several other studies have provided evidence that a racially concordant interaction can benefit medication adherence among African Americans[35]. Since the number of Caucasian physicians greatly overwhelms the number of black physicians in the US[36], this represents an additional barrier to African Americans in adhering to treatment regimens.

Diet and Exercise

As stated above, obesity is highly associated with poor glycemic control and the onset of Type 2 diabetes, and obesity rates in the African American population are higher than that of the Caucasian population. A brief discussion about the various factors that influence obesity and how they may differentially affect African Americans will be provided in this section.

Obesity is largely a product of dietary habits, physical activity levels, genetics, and age.[37] Other factors like smoking and certain prescription medications may also affect macronutrient metabolism and lead to weight gain.[37] Gender, income, education, and race are highly predictive of obesity rates within populations. Among Caucasian men, income levels are not related to obesity rates, but among African Americans and Mexican Americans, increasing income levels are associated with increasing obesity rates.[38,39] Interestingly, these trends are almost exactly reversed for women, with increasing income levels being associated with decreasing obesity rates among Caucasian women and no relationship existing between income and obesity levels among African American and Mexican American women.[38,39] Men and women of all races with college degrees have decreased obesity rates, but few trends exist for different education levels

below that of a college graduate.[38] In 2014, 22.2% of African Americans in the U.S. had a college degree, compared to 32.3% for Caucasian Americans.[40] This disparity in educational attainment may in part explain the overall increased obesity rates for African Americans. The factors that determine an individual's ability to manage their weight (genetics, age, diet, physical activity, smoking, etc.) may be related to the interactions between race, gender and income levels. The reasons for which these trends in obesity exist are beyond the scope of this thesis, but nevertheless serve to illustrate the point that race and socioeconomic status interact to influence obesity rates, which in turn contributes to the increased prevalence of diabetes in the African American population.

Additionally, African American culture surrounding food and eating habits also can negatively impact their diet. A study by Airhihenbuwa *et al.* examined attitudes towards African American cultural food practices among a population of African Americans in an urban community in Pennsylvania. The study found that many African Americans identify with the practice of eating "soul foods" like macaroni, grits, biscuits, fried chicken, chitlins, fat-back, ham hocks, fried okra, home fries, and more.[41] Interviewees attributed the development and maintenance of these practices in part to the influences of slavery, racial discrimination and socioeconomic status. While some of these foods, particularly the vegetables, are very micronutrient rich, fried foods and fatty meats are also high in fat, cholesterol and sodium. These dietary practices may contribute to the increased obesity rates in African Americans. It is thought that interventions directed at improving the dietary habits of African American ought to take these cultural practices into account.

Empowerment Theory

A promising new perspective for analyzing racial disparities in medication adherence and diabetes outcomes is provided by the theory of "patient empowerment," which was proposed by Anderson and Funnel. Anderson & Funnell (2000) advocate a self-empowerment-based model for diabetes care[42], in which patients develop a personalized plan based upon their own priorities and goals for their diabetes management, rather than providers exhorting patients to more closely adhere to their recommendations, which may or may not align with the patient's lifestyle and personal goals. The extent to which a patient has increased their self-efficacy and control over their own diabetes management relative to their goals can be measured by the Diabetes Empowerment Scale (DES).[42] This new viewpoint represents a paradigm shift in diabetes care, in which the focus on compliance with physicians' recommendation has been replaced with a focus on empowering patients to manage their own diabetes according to their own values and lifestyle.[43] The present project was especially focused on understanding how this new approach affects African American patients with diabetes.

Little research has been done on the effectiveness of empowerment-based diabetes care on African Americans specifically and on the extent to which African Americans are already empowered compared with Caucasian Americans (based on the DES). Anderson *et al.* (2005) performed a randomized control trial to examine the effectiveness of empowerment-based diabetes self-management education programs and found that the programs were able to significantly improve DES score and glycemic control.[44] Another study by Steinhardt *et al.* (2010) tested a similar intervention, called the "Diabetes Coaching Program," that was tailored for African Americans.[45] This study

also found significant improvements in DES score and glycemic control. The results of these studies suggest that empowerment-based interventions are effective in improving African Americans' diabetes care.

Although the efforts aimed at empowering African American patients with diabetes have been successful, it remains unclear as to whether there is an underlying ethnically based disparity in diabetes-specific empowerment. A study by Sarkar *et al.* (2006) found that self-efficacy did not significantly differ between African Americans and Caucasian Americans[46], although they used an alternative diabetes self-efficacy scale, rather than Anderson's DES. Wallston *et al.* (2007) used the Perceived Diabetes Self-Management Scale (PDSMS), another measure of how competent and prepared patients feel to manage their diabetes, and found that Caucasian Americans had baseline scores equal to that of African American patients with diabetes.[47] However, there have been no reports of baseline DES scores stratified by race, which makes it difficult to determine whether this factor is important in the ethnic disparity in diabetes outcomes. In this study, baseline DES scores will be compared between Caucasian Americans and African Americans in order to shed more light on the causes of these disparities.

Treatment Regimen and Comorbidities

An additional question that will be explored in this thesis is the relationship of treatment regimen and comorbidities with glycemic control, specifically whether the relationship differs by race. As mentioned previously, exogenous insulin is prescribed to individuals with Type 1 diabetes or poorly controlled Type 2 diabetes. Moreover, poorly controlled diabetes can lead to the development of various complications, which increase

the overall burden on these patients. Chronic hyperglycemia can lead to vascular, retinal and peripheral nerve damage, which may affect the ability of individuals with diabetes to exercise, take medications or perform other diabetes self-management behaviors. Additionally, individuals with diabetes often have several other preexisting conditions that did not result from their diabetes, such as depression, asthma, cardiovascular disease, obesity, and chronic obstructive pulmonary disease (COPD), which can also create difficulties in the management of diabetes.[48] Because African Americans have more poorly controlled diabetes than Caucasian Americans, one might expect that treatment with exogenous insulin and a high number of comorbid conditions are more common among African Americans. Thus, it is possible that the ethnic disparity in diabetes management in part stems from both an increased prevalence of preexisting comorbidities and a reduced ability to self-manage diabetes care alongside these conditions among African Americans. Similarly, it is also possible that African Americans and Caucasian Americans do not respond the same way to treatment with exogenous insulin. This thesis will test these hypotheses, although the lack of longitudinal data places a limit on our ability to fully understand how the time course of the development of comorbidities and diabetes impacts HbA1c and differs by race. Nevertheless, we have the ability to examine the relationship of comorbid conditions and exogenous insulin treatment with glycemic control in a race-stratified analysis.

Objectives and Specific Aims

The objective of this research is to further our understanding of the psychosocial reasons for the large ethnic disparity in diabetes outcomes. African Americans carry a

disproportionate amount of the burden of diabetes relative to Caucasian Americans, yet the causes of this disparity are not yet fully understood. There have been many successful community level interventions, but a large disparity still remains. Understanding critical areas for improvement in diabetes care of ethnic minorities can help policymakers and health care providers shape the standards of diabetes care to alleviate the burden of diabetes on individuals, the U.S. economy and the health care system. Thus, in this study we will attempt to assess whether ethnic disparities in self-management behaviors and empowerment exist and are associated with glycemic control.

One self-management behavior, medication adherence, is a highly promising candidate for intervention if it is found to be a source of ethnic disparity in diabetes care, because of all the self-management behaviors, it is probably the best determinant of glycemic control. It has also been shown to be heavily dependent on patient-provider communication. Moreover, studies described above have shown that patient-provider communication depends on racial concordance, or lack thereof, between patient and physician. However, there is still debate as to whether broader ethnicity-based differences in medication adherence exist. This study will evaluate whether or not ethnicity-based differences in medication adherence exist and whether medication adherence is associated with glycemic control.

Additionally, diet and exercise are two related self-management behaviors that may also open the door to new promising ethnically tailored diabetes care intervention. Obesity rates are higher among African Americans, and obesity is one of the strongest risk factors for diabetes onset and progression. This may explain in part the ethnic disparity in diabetes outcomes. Culturally appropriate/sensitive/relevant interventions

could be designed to improve the dietary practices and activity levels of African American patients with diabetes. This study will also assess the relationship between ethnicity, diet and exercise, as well as the relationship between diet, exercise and glycemic control.

The model of care for treatment of type 2 diabetes has shifted from a compliance-focused and physician-centered model to an empowerment-focused and patient-centered model in the last decade and a half. It is important to understand how this model shift may differentially impact patients of different ethnicities. Additionally, it is of interest to understand whether pre-intervention levels of diabetes-specific empowerment depend on race. This study will assess whether ethnic differences in empowerment levels exist and whether they are associated with glycemic control. The conceptual model in Figure 1 depicts the main hypotheses tested in this study, which are outlined below:

1. Poor glycemic control is associated with (a) lower frequency of self-management behaviors, (b) lower levels of diabetes-specific empowerment and (c) African American ethnicity.

2. The relationship between ethnicity and glycemic control is mediated by self-management behaviors, such that African American ethnicity is associated with poorer diabetes self-management behaviors, and poorer diabetes self-management behaviors is associated with higher HbA1c.

3. African American ethnicity moderates the relationships of HbA1c with (a) diabetes treatment regimen and (b) number of comorbid conditions, such that both factors are associated with increased HbA1c for African Americans but not Caucasian Americans.

Materials and Methods

The data analyzed in this study were collected by James E. Aikens, Ph. D.. and colleagues at the University of Michigan from 2003-2008 for a study entitled, "Racial differences in diabetes-depression comorbidity," funded by the National Institutes of Health. The University of Michigan Institutional Review Board granted Eli Cornblath and James Aikens, Ph. D. exemption (HUM00068928) from approval/regulation for a secondary analysis on a modified version of this data set lacking patient identifiers. The following section describes the methodology by which the data were originally collected and the data analytic approach taken for this present.

Participants

Potential participants were identified using the administrative and clinical databases of the Henry Ford Health System in Detroit, Michigan. Patients were eligible if they had Type 2 diabetes mellitus, as indicated by at least one of the following: (i) at least one hospital admission with a diabetes-related ICD-9 code (250.x, 357.2, 362.0 or 366.41); (ii) at least two outpatient visits with a diabetes-related ICD-9 code; or (iii) at least one prescription for an oral glucose-lowering medication or monitoring supplies. In order to be eligible, patients also had to be of either Caucasian or African-American ethnicity, able to complete self-report instruments and not diagnosed with bipolar depression.

Procedures

Eligible patients were mailed a study invitation letter, followed by a recruitment telephone call from research staff (including both African-American and Caucasian recruiters) for further screening and enrolment scheduling. After informed consent, participants attended a research appointment for assessment of baseline variables including diabetes regimen adherence and self-care behaviors, long-term glycemic control, empowerment, medical characteristics, and demographic characteristics. All procedures were approved by the University of Michigan Institutional Review Board.

Measures

Medication adherence was assessed using the 4-item measure developed by Morisky et al., which elicits information about the presence of various forms of medication non-adherence and has demonstrated concurrent and predictive validity and adequate internal consistency.[49] Additional self-care behaviors (diet, exercise, foot care and blood glucose monitoring) were assessed with the Summary of Diabetes Self-Care Activities (SDSCA)[50], which has adequate test–retest reliability, is sensitive to change and is correlated with other measures of the same constructs. Comorbid medical illnesses were assessed by abstracting electronic medical records using a checklist of 13 common medical illnesses used in prior primary care studies.[51,52] Glycemic control (glycated haemoglobin; HbA1c) was measured with the DCA 2000 (GMI Inc., Ramsey, MN, USA), which analyses capillary blood samples through a monoclonal antibody method. Participants classified themselves using U.S. Census racial/ethnic categories. Socio-

economic status (SES) was assessed using the U.S. Census Bureau Index of Socio-economic Status[12] adjusted for the current regional Consumer Price Index.

Data analysis

Descriptive statistics were calculated to summarize study variables. Variables with skewed distributions were rank converted for analysis. Bivariate associations were conducted to identify potential confounders using Pearson correlation and independent-samples Student's t-tests for continuous variables. Associations between HbA1c, self-management behaviors, ethnicity and various covariates were analyzed using multivariable ordinary least-squares (OLS) regression. Independent variables were entered in separate blocks corresponding to demographic control covariates, main effects, and (when applicable) interaction effects, evaluated against an alpha criterion of $p < 0.05$. All analyses were adjusted for age.

Results

Preliminary analysis of bivariate associations

We conducted a preliminary analysis to identify important factors to take into account when subsequently testing for ethnically based disparities in glycemic control. As expected, African Americans had significantly higher HbA1c values than Caucasians (7.90 ± 1.90 vs. 7.34 ± 1.38, $t(286)= -2.765$, $p=0.006$) (Table 1). Interestingly, ethnicity was not significantly related to levels of either empowerment ($t(281)= -1.082$, $p=0.280$), diet ($t(283)= -1.681$, $p=0.094$), exercise ($t(285)= -0.199$, $p=0.842$), medication adherence ($t(281)= -0.585$, $p=0.559$), duration of diabetes diagnosis ($t(263)= 1.124$, $p=0.262$), or SES ($t(285)= 0.045$, $p=0.965$) (Table 1). However, several other factors were found to be disproportionately prevalent among African Americans. A significantly greater proportion of African Americans patients were being treated with exogenous insulin than Caucasians ($t(283)= -2.154$, $p= 0.0321$). African American individuals had higher number of comorbid conditions ($t(260)= 4.096$, $p<0.001$). Additionally, African Americans were significantly younger than Caucasians (59.10 ± 8.75 vs. 54.45 ± 8.16, $t(283)= 4.625$, $p<0.001$) (Table 1).

All of the ethnicity-associated medical characteristics were also associated with HbA1c. A high number of comorbid conditions (b= -0.177, $p=0.004$) and treatment with exogenous insulin (7.446 ± 0.120 vs. 8.021 ± 0.176, $t(282)= -2.791$, $p=0.006$) were both significantly associated with higher HbA1c and were more prevalent in African Americans (Table 2). On the other hand, increasing age was associated with lower

HbA1c (b= -0.303, $p<0.001$), and Caucasians were older than African Americans on average. Additionally, men had significantly higher HbA1c than women (7.91 ± 0.15 vs. 7.37 ± 0.13, $t(285)=2.723$, $p=0.007$), although sex was balanced by race (Table 2). This preliminary analysis identified several factors associated with the *a priori* dependent variables, which were therefore considered as potential control covariates in subsequent multivariable analyses.

Primary data analysis for testing hypotheses:

Hypothesis 1. **Poor glycemic control is associated with (a) lower frequency of self-management behaviors, (b) lower levels of diabetes-specific empowerment and (c) African American ethnicity.**

Multiple regression was used to determine the relationship between poor glycemic control and diabetes self-management behaviors, diabetes-specific empowerment, and African American ethnicity. For all analyses, age was used as a control covariate. Although regimen type and comorbid conditions were associated with ethnicity and predictive of HbA1c, they were not used as control covariates because treatment with exogenous insulin and a high number of comorbid conditions are themselves reflective of disparities in glycemic control. Additionally, male sex was associated with higher HbA1c, but was not included as a control covariate because this association is not present in the general population.

Test of Hypothesis 1a. For analyses of self-management behaviors, HbA1c was the dependent variable, and diabetes self-management behaviors were tested individually as the independent variables. The self-management behaviors tested were medication adherence, diet and exercise. Lower HbA1c was significantly associated with better medication adherence (b = 0.152, p = 0.008). However, HbA1c was not associated with diet (b = -0.089, p = 0.122) or exercise (b = -0.037, p = 0.513) (Table 2).

Test of Hypothesis 1b. To test for a potential relationship between HbA1c and diabetes-specific empowerment, HbA1c was used as the dependent variable and diabetes-specific empowerment was the independent variable. Higher diabetes-specific empowerment was significantly was associated with lower HbA1c (b = -0.216, $p < 0.001$) (Table 2).

Test of Hypothesis 1c. To test for ethnically based disparities in glycemic control, HbA1c was used as the dependent variable and ethnicity was used as the independent variable. Although African Americans in this study had worse glycemic control overall (Table 1), African American ethnicity was not significantly associated with HbA1c in the presence of age as a control covariate (b = 0.098, p = 0.095), suggesting that differences in age account for ethnically based disparities in HbA1c (Table 3).

Hypothesis 2.

a. **The relationship between ethnicity and glycemic control is mediated by self-management behaviors, such that African American ethnicity is associated with poorer diabetes self-management behaviors, and poorer diabetes self-management behaviors is associated with higher HbA1c.**

b. **The relationship between ethnicity and glycemic control is mediated by diabetes-specific empowerment, such that African American ethnicity is associated with lower diabetes-specific empowerment, and lower diabetes-specific empowerment is associated with higher HbA1c.**

The results of the test of Hypothesis 1c showed that there was not a significant association between ethnicity and HbA1c. Therefore, the mediation relationship proposed in Hypothesis 2 was rejected. However, we proceeded to test the other associations specified in Hypothesis 2. Multiple regression was used to determine the relationship between ethnicity and either self-management behaviors (2a) or diabetes-specific empowerment (2b).

Test of Hypothesis 2a. For analyses of self-management behaviors, self-management behaviors (diet, medication adherence, and exercise) were individually analyzed as the dependent variable. Ethnicity was used as the

primary independent variable. Age was used as a control covariate for analyses of diet, exercise and medication adherence. Medication adherence was not significantly associated with ethnicity (b = -0.000, p = 0.999). Diet was significantly associated with ethnicity (b = 0.143, p = 0.019), with African Americans having better dietary habits than Caucasians in this study. Exercise was not associated with ethnicity (b = 0.011, p = 0.864) (Table 4).

Test of Hypothesis 2b. To test for associations between ethnicity and diabetes-specific empowerment, empowerment was used as the dependent variable. Ethnicity was used as the independent variable and age was used as a control covariate. Diabetes-specific empowerment scores did not differ significantly by ethnicity (b = 0.079, p = 0.198) (Table 4).

Hypothesis 3. **African American ethnicity moderates the relationships of diabetes treatment regimen and number of comorbid conditions with HbA1c, such that treatment with exogenous insulin and the presence of comorbid conditions lead to increased HbA1c for African Americans but not Caucasian Americans.**

a. Multiple regression was conducted separately on data from Caucasian American and African American patients to analyze the relationship between HbA1c and exogenous insulin and number of comorbid conditions. HbA1c was used as the dependent variable, age was used as a

control covariate, and either regimen type or number of comorbid conditions was used as the independent variable. For whites, treatment with exogenous insulin (b = 0.152, p = 0.094) and number of comorbid conditions (b = -0.081, p = 0.413) were not significantly associated with HbA1c (Table 5). For African Americans, treatment with exogenous insulin (b = 0.073, p = 0.341) and number of comorbid conditions (b = -0.113, p = 0.168) were not significantly associated with HbA1c (Table 5).

Discussion

The main goal of this research project was to gain a better understanding of the reasons behind ethnic disparities in Type II diabetes outcomes. Several theories exist that attempt to explain this disparity, yet it still remains unclear which factors contribute most to the disparity and by what mechanisms. A better understanding of this ethnic disparity would help health care professionals and policymakers design programs and practices that would help improve diabetes care for African Americans. In this study, three main hypotheses about the ethnic disparity were proposed and examined: 1) performance of diabetes self-management behaviors (medication adherence, diet, and physical activity) is lower among African American patients, which contributes to poor glycemic control, 2) patient empowerment is lower among African American individuals, which leads to poor diabetes management and worse glycemic control, and 3) treatment with exogenous insulin and increased comorbidities would have a larger negative impact on HbA1c for African Americans than for Caucasian Americans. Conceptual models outlining the hypotheses we developed to examine these theories are presented in Figure 1.

In order to test these hypotheses, we studied a group of 250 patients from the Henry Ford Health System, half of whom were self-identified as African American / Black and the other half as Caucasian American / White. Survey questionnaires were administered to collect data on diabetes self-management behaviors, demographic information, and social factors, while HbA1c values were obtained for each patient by blood testing. The present analysis of these data failed to confirm most of the study hypotheses surrounding ethnic disparities. Nevertheless, by indicating the unfruitfulness of certain directions, the findings certain might still be helpful in improving healthcare

professionals' understanding of ethnic disparities in diabetes care and ultimately help lead to novel interventions aimed at combating these disparities.

As expected, better medication adherence and higher diabetes-specific empowerment were associated with better glycemic control. Surprisingly, after adjusting for age, there was no significant relationship between ethnicity and HbA1c. Also, diet and exercise were not related to HbA1c. Thus, our first two hypotheses were not supported, as there was no relationship between the outcome variable and the main predictor. However, further hypothesis testing was conducted to identify potential relationships between ethnicity and mediator variables (self-management behaviors and empowerment). These tests showed that medication adherence, diabetes-specific empowerment and exercise did not significantly differ by ethnicity. Although diet was significantly healthier among African Americans, it was not related to HbA1c. Additionally, while treatment with exogenous insulin and a high number of comorbid conditions were more prevalent among African American patients and were associated with higher HbA1c overall, neither of these factors were associated with HbA1c for Caucasians or African Americans after adjusting for age. Overall, that the factors we analyzed did not significantly contribute to ethnic disparities in diabetes outcomes; nonetheless these results may help redirect efforts to reduce disparities towards other factors and lay the foundation for future studies to investigate ethnic disparities in different aspects of patient care.

Our preliminary analyses showed that there was a significant ethnic disparity in glycemic control, with African Americans having higher HbA1c than Caucasian Americans. However, after the inclusion of age as a control covariate, we found that

ethnicity was not related to glycemic control in our study, which was surprising, given that most studies of diabetes patients demonstrate vast ethnic disparities in glycemic control even after adjusting for potential confounders. In our sample, age was disproportionately represented with respect to race, in that the group of African Americans contained a constituent of younger patients, while the group of Caucasian Americans lacked younger patients but contained several older patients. In the general population, older individuals with diabetes typically have better glycemic control than younger individuals.

This imbalance in age possibly explains why there was a significant relationship between ethnicity and Hba1c before accounting for control covariates, but it does not explain the lack of disparity after statistical adjustment for patient age. Limitations inherent to the study design may explain this lack of association; namely, every patient in our sample had private health insurance, which could have acted as an SES "equalizer," masking the effects of African American ethnicity on glycemic control. If every patient had easy, affordable access to quality diabetes care, then factors that ordinarily lead to disparities may have been resolved. If this were the case, that would provide a very interesting insight into the mechanisms of ethnic disparities in diabetes outcomes. For example, it may suggest that access to health information and providers, doctor-patient communication and SES in general are important determinants of glycemic control for African Americans. It would also suggest that genetic differences are an unlikely explanation for elevated HbA1c among African Americans, because equal insurance would not have overcome any significant genetic component and we would have seen a disparity in our sample. This issue will be further discussed in the "Limitations" section.

Although this result conflicted with the predictions made by the hypotheses, we proceeded with data analysis in order to gain other insights about factors that influence glycemic control for all patients, as well as African Americans in particular.

Our second hypothesis was that medication adherence mediated the relationship between ethnicity and HbA1c. While there was no relationship between ethnicity and HbA1c in our study, we still analyzed the other aspects of this mediation relationship and found that there was an association between medication adherence and glycemic control, such that higher medication adherence was associated with better glycemic control. The literature largely supports this result, as many studies have shown that medication adherence is important for the management of chronic conditions, including diabetes. Thus, improving adherence to OHAs and injected insulin regimens remains an important target in trying to improve diabetes outcomes for the general population of individuals with diabetes. As stated in the introduction, the literature presents conflicting data as to whether ethnically based disparities in medication adherence exist. The presence or absence of a disparity likely depends on differences in the populations sampled in each of these studies. While we hypothesized that medication adherence would be poorer among African Americans, adjusted analyses revealed a lack of association between African American ethnicity and medication adherence. This finding gives further support to the idea that ethnic disparities in medication adherence do not underlie disparities in diabetes outcomes.

In addition to medication adherence, diet and exercise are two other important diabetes self-management behaviors. When carried out consistently, healthy diet and frequent exercise are considered very effective ways to manage blood sugar levels and

are recommended by health care providers before resorting to OHAs. Thus, we hypothesized that positive diet and exercise habits were associated with lower blood sugar and also performed less frequently by African Americans, serving as a source of disparity in glycemic control. Surprisingly, we found that neither diet nor exercise were significantly associated with glycemic control. One potential explanation for these results is that no patient in the study had a healthy enough diet or frequent enough exercise to effectively manage their diabetes, so small improvements in these behaviors did not translate to significant changes in HbA1c. Similarly, individuals may have filled out their questionnaire based on a "good week," rather than accurate representation of their overall exercise and diet habits. African Americans had significantly better diet habits than Caucasian Americans, but this difference is likely not important because of the lack of association between diet and HbA1c and the fact that the difference was not large in magnitude. We also found no significant difference in exercise habits by race. The lack of association with ethnicity did not align with our predictions; however, the homogeneity of SES in the sample may also explain this result, especially because diet and exercise are so highly correlated with SES. African American cultural dietary practices were discussed in the introduction and cited as a potential contributing factor to poor diet among African Americans, but it is possible that these cultural practices are not as salient among African Americans in the Northern United States as they are in the south and thus are largely not relevant to the population we studied.

Next, we examined associations between diabetes-specific empowerment and glycemic control and tested whether empowerment mediated any association between race and glycemic control. Although we already found that race was not associated with

glycemic control, we nevertheless tested the other associations specified by this mediation hypothesis in order to gain further insights into the factors that influence glycemic control. These associations were 1) empowerment and glycemic control, and 2) race and empowerment. Our results showed that higher levels of diabetes-specific empowerment were associated with better glycemic control. This finding provides support for the importance of empowerment in a patient's ability to effectively manage their diabetes. Although we looked retrospectively at the association between glycemic control and empowerment, this positive association nevertheless affirms the idea that empowerment-based treatment programs are promising candidates to improve diabetes self-management for patients regardless of race. Almost no quantitative studies have simultaneously analyzed and compared diabetes-specific empowerment, self-efficacy or other similar constructs for both African Americans and Caucasian Americans, but those studies that have showed no significant racial differences in baseline scores. However, a review of the qualitative literature led us to hypothesize that empowerment would be lower among African Americans and be a large driving factor of ethnic disparities in glycemic control. Contrary to the predictions we made prior to analysis, we did not find a significant association between empowerment and ethnicity; thus, this study supports the lack of an ethnic disparity in baseline empowerment scores. However, it is also possible that in our sample, African Americans had higher levels of empowerment than in a population without HAP insurance.

Our last hypothesis was designed to understand ethnic disparities in diabetes outcomes, as opposed to disparities in self-management behaviors. We predicted that the HbA1c was associated with treatment with exogenous insulin and comorbidities in a

race-dependent fashion, such that the presence of comorbid conditions and treatment with exogenous insulin among African Americans leads to higher HbA1c, moreso than for Caucasian Americans, if at all. After stratifying the data by ethnicity, we found that for both races, there was no association between HbA1c and treatment with exogenous insulin or number of comorbid conditions. Our data was not well suited to perform this analysis, which may limit our ability to interpret our results. Because we did not follow these patients longitudinally from their initial diabetes diagnosis, we could not monitor the development of comorbid conditions in relation to changes in HbA1c. Additionally, while we recorded whether patients were being treated with exogenous insulin at the time of the study, we did not obtain data about the time at which they began that treatment regimen, which may be a confounding factor in this analysis.

Limitations

There were several limitations inherent to the study design that may have influenced the interpretation of the data. One of the most global issues with our study was the fact that we relied heavily on self-report survey data. While biological measures such as HbA1c, BMI, and sex were included, a majority of the data was collected via questionnaires that the patients were allowed to fill out on their own. Self-report data has limited reliability for a variety of reasons. Lenzner et al. describe several elements of survey design, such as difficult vocabulary, complicated syntax and poor clarity, which can increase the burden of responding and lead to inaccurate data.[53] More specifically, self-report was among the least valid methods available to measure medication adherence.[29] Better-validated methods for measuring adherence include pill counts and

collateral reports, because these methods veer closer towards direct observation than self-report. Crowne and Marlowe (1964) introduced the theory that study participants respond to surveys in a way that presents themselves as more socially desirable.[54] In our study, this may translate into patients overestimating their medication adherence, diet and exercise. But although our data relies on self-report, the scales we used have been reasonably well-validated. For example, the Morisky Scale of Medication Adherence is used in many studies and has demonstrated high validity. Morisky *et al.*, the group that developed the scale, conducted a study in 1986 of patients being treated with antihypertensive medications, demonstrating that Morisky scores were significantly correlated with blood pressure control.[49] Additionally, they conducted a similar study testing both the 4-item scale a modified 8-item scale (not used in this thesis) in 2008, again showing that adherence scores were correlated with control of blood pressure.[55]

One of the major limitations mentioned above was the underrepresentation of individuals with lower SES. Part of the selection process for this study was to find individuals with private insurance from the Michigan Health Alliance Plan (HAP). Thus, health care access did not vary widely in our sample. Also, income and education levels were relatively homogeneous within our sample as well. This bias may have skewed our data our masked disparities that were largely due to race-related disparities in SES. Large-scale studies have reported significantly increased prevalence of diabetes among lower SES populations[56] and have shown that diabetes is more poorly controlled in lower SES individuals[57], but our study failed to find an association between SES and HbA1c, likely due to the fact that SES was homogeneous in our study. This lack of association prevented us from analyzing the interaction between SES and race or other factors.

Nevertheless, we had enough of a representative range for factors like medication adherence and empowerment that allowed us to identify important associations regardless.

Another limitation was the lack of data on physician race and a lack of minority physicians. There was only a small number of physicians whom patients identified as non-Caucasian (n = 6), which was insufficient for any analysis of the role that racial discordance might play in diabetes care. Additionally, because data on the race of the physician was collected by patient self-report, the races reported may not align with how the physicians would identify their own race. Obtaining further data on the race of the physician treating each patient would greatly enhance our ability to ask the important question of whether racial concordance influences the quality of diabetes care. Because we did not find any factors that were significant predictors of ethnic disparity in glycemic control and diabetes care, it would have been interesting to explore doctor-patient communication as a potential reason. A 2008 survey by the Center for Studying Health System Change found that 73.7% of physicians identified as non-Hispanic White, while only 3.8% of physicians identified as non-Hispanic Black[36], which is consistent with the lack of minority physicians in our study without intentional recruitment. It may have been necessary to design the study so that the sample was balanced with respect to each combination of patient race and physician race, i.e. Caucasian patients with African American physicians, Caucasian patients with Caucasian physicians, African American patients with Caucasian physicians, and African American patients with African American physicians. Moreover, additional survey questionnaires could have been added to assess how these patients perceived the communication. However, the existing tools

for measuring the extent to which a patient feels that a physician has communicated effectively and provided patient-centered care are limited in their consistency and reliability.[58,59]

Finally, our heavy reliance on quantitative data may have oversimplified and masked phenomena that explain the ethnic disparity in diabetes outcomes. Inclusion of in-depth, qualitative interviews could have helped capture the deeper, more personal aspects of diabetes care and would have allowed us to triangulate our findings. Qualitative interview data would have particularly provided us with a richer perspective on each patient's experience communicating with health care professionals and how that impacted their ability to manage their diabetes. It would also give us more insights about the implications of holding an African American racial identity in a medical setting, both for physicians and patients, which is important for a field that has historically been dominated by Caucasian American males.[36]

Implications

Type 2 Diabetes Mellitus is one of the most common chronic diseases, afflicting 8% of the US population. Prevalence of diabetes has increased greatly over the last 50 years, and even the last 10, generating an enormous social, economic and medical burden on the US. Thus, it is an urgent medical concern to help reduce the development of and treat diabetes. The burden of diabetes is not shared equally among the US population; African American individuals comprise a disproportionate amount of individuals with diabetes and experience more diabetes-related complications that individuals with diabetes of other ethnic backgrounds. This thesis presents an overview of research aimed

at identifying the reasons behind ethnic disparities in diabetes, with a focus on psychosocial and behavioral explanations, followed by novel research on how ethnicity, medication adherence, patient empowerment and treatment regimens relate to management of chronic hyperglycemia.

The findings in this study point towards several potential actions that can be taken to both improve the ethnic disparity and further understand it. While this study did not directly identify factors that differed by race and were associated with glycemic control, it still reaffirmed current perspectives on diabetes treatment and highlights the need for further studies on diabetes in African Americans. Diabetes-specific empowerment was associated with better glycemic control, which provides support for the continuation and expansion of the empowerment approach to diabetes care. This study compared baseline levels of empowerment between African American and Caucasian American individuals and showed that they were equal, a comparison that has rarely been made in the literature. While the empowerment approach has been shown to be successful among African Americans, it may not be a specific way to address the ethnic disparities in diabetes outcomes. Moreover, it is important to thoroughly examine how the empowerment-based approach might differentially impact Caucasian and African American patients when implemented on a larger scale with a more diverse patient population. The effectiveness of empowerment-based programs may differ by SES, occupation, personality type, education levels, various lifestyle differences, and more. Health care providers could begin to adopt empowerment-based programs as their standard protocol for diabetes treatments and the government could create financial incentives for institutions that instate these programs. If an increased number of

empowerment-based programs could effectively reduce the economic burden of diabetes, then the financial incentive would be reason enough alone to include empowerment-based programs in diabetes treatment.

Medication adherence was also associated with better glycemic control. Although the empowerment model deemphasizes the concept of "adherence," this finding nevertheless supports the idea that taking medications as prescribed does lead to better glycemic control. Interestingly, medication adherence did not differ by race, which may suggest that ethnic disparities do not manifest themselves through disparities in medication adherence. Similarly, improving medication adherence will also likely improve diabetes management, but as Anderson and others have suggested, an adherence-centered model of diabetes care may not be effective.

Although this study did not examine any physiological or genetic data, our results point towards a need for more biological research related to ethnic disparities in diabetes. Diet and exercise were not associated with HbA1c, although African Americans reported slightly healthier diet than Caucasians. Biological studies could be done to identify ethnic differences in metabolism and response to improvements in diet and physical activity levels. Moreover, if the behavioral and psychosocial factors related to glycemic control truly did not differ by ethnicity, then genetic differences may indeed play a role in this disparity. Further research into potential genetic factors responsible for increased risk for Type 2 diabetes and increased rates of diabetes-related complications among African Americans could lead to the development of new medications and other treatments to more effectively compensate for these differences.

These results could also inform the design of a randomized controlled trial to investigate the role of other factors in ethnic disparities in diabetes care. Our lack of variation in SES underscores the importance of having a wide representation of SES levels in subsequent studies. Ethnicity may interact with SES in ways that we could not assess in our data, and this analysis should be conducted before investigating other perspectives. One important factor that remains to be investigated is the role of doctor-patient communication. It would be useful to narrow down the focus from medication adherence and empowerment to the factors that directly influence an individual's ability to take medicine as prescribed, and the extent to which African American patients become empowered. Communication with health care providers plays a large role in improving patient empowerment and in promoting medication adherence. Lack of racial concordance can have a negative impact on patient physician communication and subsequent medication adherence[35]; thus, a randomized controlled trial could be designed to more directly examine how the African American racial identity of patient can affect how they are perceived and treated in a health care setting. Studies such as these would be helpful in educating physicians and other health care providers to have more successful encounters with patients from diverse populations.

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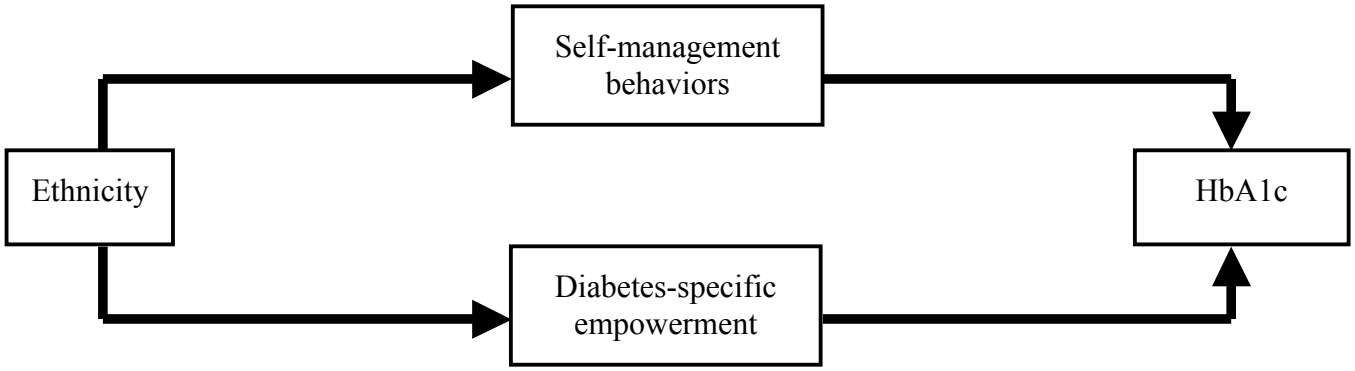
Figure 1. Conceptual models of main hypotheses.

Hypotheses 1 and 2:



Hypothesis 3:

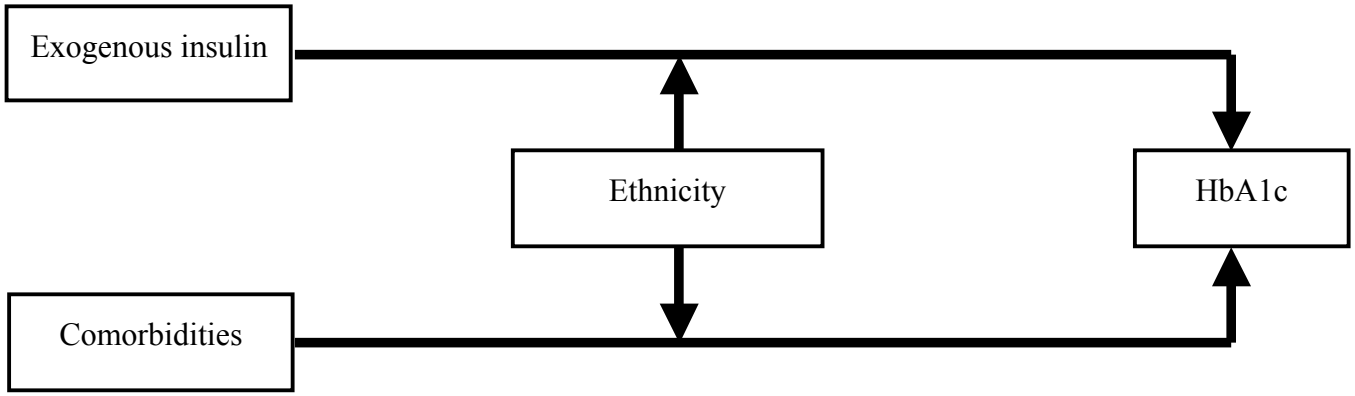


Table 1. Patient demographics and baseline medical characteristics.

Variable	Total sample (n=288)	White (n=124)	Black (n=164)	p value
Age, mean ± SD	56.47 ± 8.72	59.10 ± 8.75	54.45 ± 8.16	**<0.001**
Female, %	48.08	48.38	47.85	0.929
Black, %	57.09	NA	NA	NA
Insulin and oral meds, %	39.65	32.52	45.06	**0.032**
Diabetes duration[i], mean ± SD	10.73 ± 7.95	11.36 ± 9.42	10.25 ± 6.62	0.262
SES[ii], mean ± SD	64.83 ± 17.74	64.89 ± 17.33	64.80 ± 18.10	0.965
HbA1c, mean ± SD	7.66 ± 1.72	7.34 ± 1.38	7.90 ± 1.90	**0.006**
Medication adherence[iii], mean± SD	4.80 ± 1.01	4.76 ± 0.93	4.83 ± 1.08	0.559
Empowerment[iv], mean ± SD	3.69 ± 0.69	3.63 ± 0.71	3.72 ± 0.67	0.280
Comorbid conditions[v], mean ± SD	0.79 ± 0.93	1.04 ± 1.04	0.58 ± 0.77	**<0.001**
Diet, mean ± SD	3.69 ± 1.62	3.51 ± 1.76	3.83 ± 1.49	0.094
Exercise, mean ± SD	2.20 ± 1.95	2.18 ± 2.06	2.22 ± 1.88	0.842

[i] Total n=265, White n=114, Black n=151.

[ii] Hollingshead SES Index.

[iii] Morisky Medication Adherence Scale[49]. Scores range from 4 to 8, higher score indicates poorer medication adherence.

[iv] Diabetes Empowerment Scale[42]. Scale of diabetes-related psychosocial self-efficacy.

[v] Based on self-reported hypertension, cardiovascular disease, hyperlipidemia, cancer, stroke, arthritis, chronic lung disease, migraine, asthma, and low back pain. Total n=262, White n=118, Black n=144.

Table 2. Table of bivariate associations between outcome variable and potential control covariates.

Potential control covariate	HbA1c[i]	p value
Age	-0.303	**<0.001**
Diabetes duration[ii]	0.042	0.494
SES[iii]	-0.032	0.589
Medication adherence[iv]	0.203	**0.001**
Empowerment[v]	-0.240	**<0.001**
Comorbid conditions[vi]	-0.177	**0.004**
Diet	-0.145	**0.014**
Exercise	-0.062	0.296
OHA only – Insulin and OHA, mean difference ± SD	-0.575 ± 0.206	**0.006**
Male - Female, mean difference ± SD	0.544 ± 0.200	**0.007**
White - Black, mean difference ± SD	-0.559 ± 0.202	**0.006**

[i] Pearson correlation is reported in this column unless otherwise specified in "Potential Control Covariate" column.

[ii] Total n=265, White n=114, Black n=151.

[iii] Hollingshead SES Index.

[iv] Morisky Medication Adherence Scale[49]. Scores range from 4 to 8, higher score indicates poorer medication adherence.

[v] Diabetes Empowerment Scale[42].

[vi] Based on self-reported hypertension, cardiovascular disease, hyperlipidemia, cancer, stroke, arthritis, chronic lung disease, migraine, asthma, and low back pain. Total n=262, White n=118, Black n=144.

Table 3. Multivariable OLS regression analyses of HbA1c.

Dependent Variable	Independent Variables	Standard β	p value
n= 285	Age	-0.277	**<0.001**
R^2 total= 0.100	Ethnicity[i]	0.098	0.095
P < 0.001			
n= 280	Age	-0.307	**<0.001**
R^2 total= 0.132	Medication Adherence[ii]	0.152	**0.008**
P < 0.001			
n= 282	Age	-0.291	**<0.001**
R^2 total= 0.0946	Diet	-0.089	0.122
P < 0.001			
n= 280	Age	-0.304	**<0.001**
R^2 total= 0.0948	Exercise	-0.037	0.513
P < 0.001			
n= 280	Age	-0.300	**<0.001**
R^2 total= 0.146	Empowerment	-0.216	**<0.001**
P < 0.001			

[i] Coded 0 for white, 1 for African American.

[ii] Morisky medication adherence scale[49]. Higher scores indicate lower adherence.

Table 4. Multivariable regression analyses of self-management behaviors and empowerment.

Dependent Variable	Independent Variables	Standard β	p value
Medication Adherence	Age	-0.155	**0.012**
n= 280	Ethnicity[i]	-0.000	0.999
R^2 total= 0.024			
P = 0.034			
Diet	Age	0.198	**0.001**
n= 282	Ethnicity	0.143	**0.019**
R^2 total= 0.044			
P = 0.002			
Exercise	Age	0.045	0.468
n= 284	Ethnicity	0.011	0.864
R^2 total= 0.002			
P = 0.768			
Empowerment	Age	0.088	0.154
n= 280	Ethnicity	0.079	0.198
R^2 total= 0.011			
P = 0.225			

[i] Coded 0 for white, 1 for African American.

Table 5. Multivariable regression analyses of HbA1c on regimen type by ethnicity.

Ethnicity	Independent Variables	Standard β	p value
White	Age	-0.190	**0.037**
n= 123	Regimen Type[i]	0.152	0.094
R^2 total= 0.050			
P = 0.046			
African American	Age	-0.349	**<0.001**
n= 158	Regimen Type	0.073	0.341
R^2 total= 0.138			
P < 0.001			
White	Age	-0.141	0.157
n= 118	Comorbid Conditions	-0.081	0.413
R^2 total= 0.035			
P = 0.127			
African American	Age	-0.293	**<0.001**
n= 140	Comorbid Conditions	-0.113	0.168
R^2 total= 0.109			
P < 0.001			

[i] Coded 0 for OHAs only, 1 for OHAs and exogenous insulin.